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=> s kato shigeaki /au

L1 448 KATO SHIGEAKI

=> s takeyama ken-ichi /au

L2 53 TAKEYAMA KEN-ICHI

=> s kitanaka sachiko /au

L3 40 KITANAKA SACHIKO

=> s 11 and 12 and 13

L4 26 L1 AND L2 AND L3

=> dup rem 14

PROCESSING COMPLETED FOR L4

L5 14 DUP REM L4 (12 DUPLICATES REMOVED)

=> d 15 total ibib

L5 ANSWER 1 OF 14 MEDLINE DUPLICATE 1

ACCESSION NUMBER: 2002266794 MEDLINE

DOCUMENT NUMBER: 22001224 PubMed ID: 12006701

TITLE: Molecular genetics of vitamin D- dependent hereditary

rickets.

AUTHOR: Kato Shigeaki; Yoshizazawa Tatsuya; Kitanaka Sachiko; Murayama Akiko; Takeyama Ken-ichi

CORPORATE SOURCE: Institute of Molecular and Cellular Biosciences, University

of Tokyo, and CREST, Japan Science and Technology

Corporation, Saitama, Japan.. uskato@mail.ecc.u-tokyo.ac.jp

SOURCE: HORMONE RESEARCH, (2002) 57 (3-4) 73-8. Ref: 39

Journal code: 0366126. ISSN: 0301-0163.

PUB. COUNTRY: Switzerland

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 200210

ENTRY DATE: Entered STN: 20020514

Last Updated on STN: 20021031 Entered Medline: 20021030

L5 ANSWER 2 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE

2

ACCESSION NUMBER: 2001:476798 BIOSIS

PREV200100476798 DOCUMENT NUMBER: The molecular basis of vitamin D-dependent rickets type I. TITLE: AUTHOR(S): Kitanaka, Sachiko (1); Takeyama, Ken-ichi ; Murayama, Akiko; Kato, Shigeaki CORPORATE SOURCE: (1) Department of Pediatrics, Faculty of Medicine, The University of Tokyo, 7-3-1, Bunkyo-ku, Tokyo, 113-8655 Japan Endocrine Journal, (August, 2001) Vol. 48, No. 4, pp. SOURCE: 427-432. print. ISSN: 0918-8959. DOCUMENT TYPE: General Review LANGUAGE: English SUMMARY LANGUAGE: English ANSWER 3 OF 14 CAPLUS COPYRIGHT 2003 ACS ACCESSION NUMBER: 1999:96372 CAPLUS DOCUMENT NUMBER: 130:163951 TITLE: Cloning of cDNA for ligand-converting enzymes from mice and human, and methods of screening nuclear receptors-binding ligands or transcription factors INVENTOR(S): Kato, Shigeaki; Takeyama, Ken-ichi ; Kitanaka, Sachiko Chuqai Seiyaku Kabushiki Kaisha, Japan PATENT ASSIGNEE(S): PCT Int. Appl., 66 pp. SOURCE: CODEN: PIXXD2 DOCUMENT TYPE: Patent Japanese LANGUAGE: FAMILY ACC. NUM. COUNT: PATENT INFORMATION: PATENT NO. KIND DATE APPLICATION NO. DATE -----______ WO 9905292 WO 1998-JP3280 19980722 A1 19990204 W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, KE, KG, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG AU 9883564 **A1** 19990216 AU 1998-83564 19980722 19990518 JP 1998-206786 JP 11127871 **A2** 19980722 EP 1024193 A1 20000802 EP 1998-933895 19980722 AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI PRIORITY APPLN. INFO.: JP 1997-212624 A 19970722 W 19980722 WO 1998-JP3280 REFERENCE COUNT: THERE ARE 14 CITED REFERENCES AVAILABLE FOR THIS 14 RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT ANSWER 4 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE L5 2000:18296 BIOSIS ACCESSION NUMBER: DOCUMENT NUMBER: PREV200000018296 No enzyme activity of 25-hydroxyvitamin D3 TITLE: lalpha-hydroxylase gene product in pseudovitamin D deficiency rickets, including that with mild clinical manifestation. Kitanaka, Sachiko; Murayama, Akiko; Sakaki, AUTHOR (S): Toshiyuki; Inouye, Kuniyo; Seino, Yoshiki; Fukumoto, Seiji; Shima, Masaaki; Yukizane, Shigenori; Takayanagi, Masaki;

Niimi, Hiroo; Takeyama, Ken-Ichi; Kato,

(1) Institute of Molecular and Cellular Biosciences,

Shigeaki (1)

CORPORATE SOURCE:

University of Tokyo, 1-1-1 Yayoi, Bunkyo-ku, Tokyo,

113-0032 Japan

Journal of Clinical Endocrinology & Metabolism, (Nov., SOURCE:

1999) Vol. 84, No. 11, pp. 4111-4117.

ISSN: 0021-972X.

DOCUMENT TYPE:

Article

LANGUAGE:

English

SUMMARY LANGUAGE:

English

ANSWER 5 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC. DUPLICATE **L5**

ACCESSION NUMBER:

1999:237495 BIOSIS

DOCUMENT NUMBER:

PREV199900237495

TITLE:

Positive and negative regulations of the renal

25-hydroxyvitamin D3 1alpha-hydroxylase gene by parathyroid

hormone, calcitonin, and lalpha, 25 (OH) 2D3 in intact

animals.

AUTHOR (S):

Murayama, Akiko; Takeyama, Ken-ichi;

Kitanaka, Sachiko; Kodera, Yasuo; Kawaguchi, Yoshindo; Hosoya, Tatsuo; Kato, Shigeaki (1)

CORPORATE SOURCE:

(1) Institute of Molecular and Cellular Biosciences,

University of Tokyo, Yayoi 1-1-1, Bunkyo-ku, Tokyo,

113-0032 Japan

SOURCE:

Endocrinology, (May, 1999) Vol. 140, No. 5, pp. 2224-2231.

ISSN: 0013-7227.

DOCUMENT TYPE:

Article

LANGUAGE:

English

SUMMARY LANGUAGE: English

L5 ANSWER 6 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE

ACCESSION NUMBER:

1999:96871 BIOSIS

DOCUMENT NUMBER:

PREV199900096871

TITLE:

Selective interaction of vitamin D receptor with transcriptional coactivators by a vitamin D analog.

AUTHOR (S):

Takeyama, Ken-Ichi; Masuhiro, Yoshikazu; Fuse, Hiroaki; Endoh, Hideki; Murayama, Akiko; Kitanaka, Sachiko; Suzawa, Miyuki; Yanagisawa, Junn; Kato,

Shigeaki (1)

CORPORATE SOURCE: (1) Inst. Molecular Cellular Biosciences, Univ. Tokyo,

Yayoi 1-1-1, Bunkyo-ku, Tokyo 113 Japan

SOURCE:

Molecular and Cellular Biology, (Feb., 1999) Vol. 19, No.

2, pp. 1049-1055.

ISSN: 0270-7306.

DOCUMENT TYPE:

Article

LANGUAGE:

English

ANSWER 7 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE L5

ACCESSION NUMBER:

2000:1847 BIOSIS

DOCUMENT NUMBER:

AUTHOR (S):

PREV20000001847

TITLE:

Enzymatic properties of human 25-hydroxyvitamin D3 lalpha-hydroxylase. Coexpression with adrenodoxin and

NADPH-adrenodoxin reductase in Escherichia coli. Sawada, Natsumi; Sakaki, Toshiyuki; Kitanaka,

Sachiko; Takeyama, Ken-ichi; Kato,

Shigeaki; Inouye, Kuniyo (1)

CORPORATE SOURCE:

(1) Division of Applied Life Sciences, Graduate School of Agriculture, Kyoto University, Sakyo-ku, Kyoto, 606-8502

Japan

SOURCE: European Journal of Biochemistry, (Nov., 1999) Vol. 265,

No. 3, pp. 950-956.

ISSN: 0014-2956.

DOCUMENT TYPE:

Article

LANGUAGE:

English

SUMMARY LANGUAGE: English

L5 · ANSWER 8 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

1999:529546 BIOSIS ACCESSION NUMBER: DOCUMENT NUMBER: PREV199900529546

Identification of a novel vitamin D negative response TITLE:

element in the human 25-hydroxyvitamin D3

1alpha-hydroxylase gene promoter.

Murayama, Akiko (1); Takeyama, Ken-ichi (1); AUTHOR (S):

Kitanaka, Sachiko (1); Kawaguchi, Yoshindo; Hosoya,

Tatsuo: Kato, Shiqeaki (1)

(1) Institute of Molecular and Cellular Biosciences, CORPORATE SOURCE:

University of Tokyo, Tokyo Japan

Journal of the American Society of Nephrology, (Sept., SOURCE:

1999) Vol. 10, No. PROGRAM AND ABSTR. ISSUE, pp. 622A. Meeting Info.: 32nd Annual Meeting of the American Society of Nephrology Miami Beach, Florida, USA November 1-8, 1999

American Society of Nephrology

. ISSN: 1046-6673.

DOCUMENT TYPE:

Conference LANGUAGE: English

ANSWER 9 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE

1999:396754 BIOSIS ACCESSION NUMBER: PREV199900396754 DOCUMENT NUMBER:

In vivo function of VDR in gene expression-VDR knock-out TITLE:

mice.

Kato, Shigeaki (1); Takeyama, Ken-ichi; AUTHOR(S):

Kitanaka, Sachiko; Murayama, Akiko; Sekine,

Keisuke; Yoshizawa, Tatsuya

CORPORATE SOURCE: (1) Institute of Molecular and Cellular Biosciences, The

University of Tokyo, Bunkyo-ku, Tokyo, 113 Japan

Journal of Steroid Biochemistry and Molecular Biology, SOURCE:

(April June, 1999) Vol. 69, No. 1-6, pp. 247-251.

ISSN: 0960-0760.

DOCUMENT TYPE:

General Review

LANGUAGE:

English SUMMARY LANGUAGE: English

ANSWER 10 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: DOCUMENT NUMBER:

1999:431087 BIOSIS PREV199900431087

TITLE:

Identification of a novel vitamin D negative response

element in the human 25-hydroxyvitamin D3

1alpha-hydroxylase gene promoter.

Murayama, Akiko (1); Takeyama, Ken-ichi (1); AUTHOR (S):

Kitanaka, Sachiko (1); Kodera, Yasuo (1);

Kato, Shigeaki (1)

CORPORATE SOURCE: (1) Institution of Molecular and Cellular Biosciences,

University of Tokyo, Bunkyo-ku, Tokyo Japan

Journal of Bone and Mineral Research, (Sept., 1999) Vol. SOURCE:

14, No. SUPPL. 1, pp. S167.

Meeting Info.: Twenty-First Annual Meeting of the American Society for Bone and Mineral Research St. Louis, Missouri, USA September 30-October 4, 1999 American Society for Bone

and Mineral Research . ISSN: 0884-0431.

DOCUMENT TYPE:

Conference

LANGUAGE: English

ANSWER 11 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE

ACCESSION NUMBER: 1998:165815 BIOSIS DOCUMENT NUMBER: PREV199800165815

Inactivating mutations in the 25-hydroxyvitamin D3 TITLE:

lalpha-hydroxylase gene in patients with pseudovitamin

D-deficiency rickets.

Kitanaka, Sachiko; Takeyama, Ken-Ichi; AUTHOR (S):

Murayama, Akiko; Sato, Takashi; Okumura, Katsuzumi; Nogami,

Masahiro; Haseqawa, Yukihiro; Nimi, Hiroo; Yanagisawa,

Junn; Tanaka, Toshiaki; Kato, Shigeaki (1)

(1) Inst. Mol. Cell. Biosci., Univ. Tokyo, Yayoi, CORPORATE SOURCE:

Bunkyo-ku, Tokyo 113-0032 Japan

New England Journal of Medicine, (March 5, 1998) Vol. 338, SOURCE:

No. 10, pp. 653-661.

ISSN: 0028-4793.

DOCUMENT TYPE: LANGUAGE:

Article English

ANSWER 12 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE L5

1998:404828 BIOSIS ACCESSION NUMBER: PREV199800404828

DOCUMENT NUMBER:

TITLE: The promoter of the human 25-hydroxyvitamin D3

lalpha-hydroxylase gene confers positive and negative responsiveness to PTH, calcitonin, and lalpha, 25 (OH) 2D3.

Murayama, Akiko (1); Takeyama, Ken-Ichi (1); AUTHOR (S):

Kitanaka, Sachiko (1); Kodera, Yasuo (1); Hosoya,

Tatsuo; Kato, Shigeaki

(1) Inst. Mol. Cell. Biosci., Univ. Tokyo, Yayoi 1-1, CORPORATE SOURCE:

Bunkyo, Tokyo 113-0032 Japan

Biochemical and Biophysical Research Communications, (Aug. SOURCE:

10, 1998) Vol. 249, No. 1, pp. 11-16.

ISSN: 0006-291X.

DOCUMENT TYPE: LANGUAGE:

Article English

ANSWER 13 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE L5

1998:47702 BIOSIS ACCESSION NUMBER: PREV199800047702 DOCUMENT NUMBER:

A new compound heterozygous mutation in the TITLE:

11beta-hydroxysteroid dehydrogenase type 2 gene in a case

of apparent mineralocorticoid excess.

Kitanaka, Sachiko (1); Katsumata, Noriyuki; AUTHOR (S):

Tanae, Ayako; Hibi, Itsuro; Takeyama, Ken-Ichi; Fuse, Hiroaki; Kato, Shigeaki; Tanaka, Toshiaki

(1) Inst. Mol. Cell. Biosci., Univ. Tokyo, 1-1-1 Yayoi, CORPORATE SOURCE:

Bunkyo-ku, Tokyo 113 Japan

Journal of Clinical Endocrinology & Metabolism, (Dec., SOURCE:

1997) Vol. 82, No. 12, pp. 4054-4058.

ISSN: 0021-972X.

DOCUMENT TYPE:

LANGUAGE:

Article English

ANSWER 14 OF 14 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.DUPLICATE

11

1997:448293 BIOSIS ACCESSION NUMBER: DOCUMENT NUMBER: PREV199799747496

25-Hydroxyvitamin D-3 1-alpha-hydroxylase and vitamin D TITLE:

synthesis.

Takeyama, Ken-Ichi; Kitanaka, Sachiko; AUTHOR (S):

Sato, Takashi; Kobori, Masato; Yanagisawa, Junn; Kato,

Shigeaki (1)

(1) Inst. Molecular Cellular Biosciences, Univ. Tokyo, CORPORATE SOURCE:

Yayoi, Bunkyo-ku, Tokyo 113 Japan

Science (Washington D C), (1997) Vol. 277, No. 5333, pp. SOURCE:

1827-1830.

ISSN: 0036-8075.

DOCUMENT TYPE:

Article English

LANGUAGE: Engli

=> s ligand (s) precusor (s) nuclear (s) receptor

L6 1 LIGAND (S) PRECUSOR (S) NUCLEAR (S) RECEPTOR

=> s ligand (s) precursor (s) nuclear (s) receptor

L7 324 LIGAND (S) PRECURSOR (S) NUCLEAR (S) RECEPTOR

=> s ligand (s) precursor (s) nuclear (s) receptor (s) vitamin

L8 36 LIGAND (S) PRECURSOR (S) NUCLEAR (S) RECEPTOR (S) VITAMIN

=> dup rem 18

PROCESSING COMPLETED FOR L8

L9 13 DUP REM L8 (23 DUPLICATES REMOVED)

=> d 19 total ibib kwic

L9 ANSWER 1 OF 13 MEDLINE

DUPLICATE 1

ACCESSION NUMBER: 2002613512 DOCUMENT NUMBER: 22257731

2002613512 IN-PROCESS 22257731 PubMed ID: 12370121

TITLE:

The murine gene encoding parathyroid hormone: genomic organization, nucleotide sequence and transcriptional

regulation.

AUTHOR:

He B; Tong T K; Hiou-Tim F F-T; Al-Akad B; Kronenberg H M;

Karaplis A C

CORPORATE SOURCE:

Department of Medicine, SMBD-Jewish General Hospital, Lady Davis Institute for Medical Research, McGill University, 3755 Cote Ste-Catherine Road, Montreal, Quebec H3T 1E2,

Canada.

SOURCE:

JOURNAL OF MOLECULAR ENDOCRINOLOGY, (2002 Oct) 29 (2)

193-203.

Journal code: 8902617. ISSN: 0952-5041.

PUB. COUNTRY:

England: United Kingdom

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE:

English

FILE SEGMENT:

IN-PROCESS; NONINDEXED; Priority Journals

ENTRY DATE:

Entered STN: 20021010

Last Updated on STN: 20021213

The type 1 parathyroid hormone receptor (PTHR1) binds, with AB equal affinity, two ligands with distinct biological functions: PTH, the major peptide hormone controlling calcium homeostasis, and the paracrine factor, PTH-related peptide (PTHrP), a local regulator of cellular proliferation and differentiation. To clarify the complexity of possible interactions between two distinct ligands, PTH and PTHrP, and their common receptor in the intact organism, and to identify as yet unrecognized roles for PTH in normal physiology, we have cloned and. . . and, analogous to the human PTH gene, it is interrupted by two introns. The deduced mRNA encodes the 115-amino acid precursor, preproPTH. Comparison of the murine preproPTH sequence with other mammalian forms of the protein shows it to be highly conserved. share limited structural similarity to PTHrP at the amino-terminal region, a domain critical for binding and activation of their common receptor. Putative binding motifs for the transcription factors sex-determining region Y gene product, transcriptional repressor CDP, hepatic nuclear factor 3beta, GATA-binding factor 1, glucocorticoid receptor, SRY-related high mobility group box protein 5 and cAMP response element binding protein were identified in the 5' flanking region. . . Pth gene. When placed upstream of a reporter gene, these sequences failed to confer transcriptional regulation in response to 1,25(OH)(2) vitamin D(3), but responded positively to the addition of isoproterenol and forskolin. Mutational analysis identified a cAMP-response element in the Pth.

L9 ANSWER 2 OF 13 MEDLINE DUPLICATE 2

ACCESSION NUMBER:

2001684330 MEDLINE

DOCUMENT NUMBER:

CORPORATE SOURCE:

21586214 PubMed ID: 11729302

TITLE:

Nuclear receptors and lipid physiology: opening the

Y-filec

AUTHOR:

Chawla A; Repa J J; Evans R M; Mangelsdorf D J Howard Hughes Medical Institute, Gene Expression

Laboratory, The Salk Institute for Biological Studies, Post

Office Box 85800, San Diego, CA 92186-5800, USA.

SOURCE:

SCIENCE, (2001 Nov 30) 294 (5548) 1866-70. Ref: 69

Journal code: 0404511. ISSN: 0036-8075.

PUB. COUNTRY:

United States

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LANGUAGE:

English

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

200112

ENTRY DATE:

Entered STN: 20011204

Last Updated on STN: 20021227 Entered Medline: 20011231

AB Cholesterol, fatty acids, fat-soluble **vitamins**, and other lipids present in our diets are not only nutritionally important but serve as **precursors** for **ligands** that bind to **receptors**

in the nucleus. To become biologically active, these lipids must first be absorbed by the intestine and transformed by metabolic. . . regarding the mechanisms that govern these pathways. Specifically, what is the nature of communication between these bioactive lipids and their receptors, binding proteins, transporters, and metabolizing enzymes that links them physiologically and speaks to a higher level of metabolic control? Some general principles that govern the actions of this class of bioactive lipids and their nuclear receptors are considered here, and the scheme that emerges reveals a complex

molecular script at work.

ANSWER 3 OF 13 MEDLINE

DUPLICATE 3

ACCESSION NUMBER:

2001563418 MEDLINE

DOCUMENT NUMBER:

21521418 PubMed ID: 11641059

TITLE:

Regulation of human profilaggrin promoter activity in

cultured epithelial cells by retinoic acid and

glucocorticoids.

AUTHOR:

Presland R B; Tomic-Canic M; Lewis S P; Dale B A

CORPORATE SOURCE:

Department of Oral Biology, University of Washington, Box 357132, Seattle, WA 98195-7132, USA.. rp@u.washington.edu

CONTRACT NUMBER:

AR45974 (NIAMS)

P01 AM 21557 (NIADDK) R37 DE 04660 (NIDCR)

SOURCE:

JOURNAL OF DERMATOLOGICAL SCIENCE, (2001 Nov) 27 (3)

192-205.

Journal code: 9011485. ISSN: 0923-1811.

PUB. COUNTRY:

Ireland

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE:

English

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

200112

ENTRY DATE:

Entered STN: 20011022

Last Updated on STN: 20020122 Entered Medline: 20011205

AB Vitamin A and other retinoids profoundly inhibit both morphological and biochemical aspects of epidermal differentiation in vitro. Profilaggrin, like most other. . . the activity of endoproteases that convert profilaggrin to filaggrin. Profilaggrin is an abundant component of keratohyalin granules and forms the precursor of filaggrin, the keratin associated protein of the stratum corneum. In this report, we identify a region of the human. . . reporter gene were

prepared and analyzed by transfection into Hela cells and keratinocytes. We also cotransfected vectors expressing retinoic acid receptor and cultured the transfected cells in the presence and absence of ligand. The region responsive to retinoic acid was localized to a 53 bp sequence between -1109 and -1056 (relative to the. . . a cluster of five retinoic acid response elements with variable spacing and orientation. In vitro gel shift analysis demonstrated that nuclear retinoid receptors do not bind directly to the identified sequence, suggesting that the mode of regulation by RA may be indirect or that binding requires another cofactor in addition to retinoid receptors. Whereas in keratin genes retinoic acid and glucocorticoid responsive sequences frequently coincide, the glucocorticoid response element in the profilaggrin promoter. . .

L9 ANSWER 4 OF 13 BIOSIS COPYRIGHT 2003 BIOLOGICAL ABSTRACTS INC.

ACCESSION NUMBER: 2002:129801 BIOSIS DOCUMENT NUMBER: PREV200200129801

TITLE: X-retinoic acid receptor a fusion genes in acute

promyelocytic leukemia interfere with retinoid and

peroxisome-proliferator signaling pathways.

AUTHOR(S): Hamadani, Soheila A. (1); Zhang, Tong; Dorrell, Craig;

Dick, John; Wells, Richard; Kamel-Reid, Suzanne

CORPORATE SOURCE: (1) Laboratory Medicine and Pathobiology and Molecular and Medical Genetics, Institute of Medical Science, University

of Toronto, Toronto, ON Canada

SOURCE: Blood, (November 16, 2001) Vol. 98, No. 11 Part 1, pp. 88a.

http://www.bloodjournal.org/. print.

Meeting Info.: 43rd Annual Meeting of the American Society of Hematology, Part 1 Orlando, Florida, USA December 07-11,

2001

ISSN: 0006-4971.

DOCUMENT TYPE: Conference LANGUAGE: English

English Acute promyelocytic-leukemia (APL) is characterized by selected expansion of immature myeloid precursors that are blocked at the promyelocytic stage of development. In APL, five different fusion partners of RARalpha have been identified, these include the promyelocytic leukemia gene (PML), the promyelocytic leukemia zine finger gene (PLZF), nucleophosmin (NPM), nuclear mitotic apparatus (NuMA), and the Stat5b gene. The fact that all APL subtypes with various RAR fusion partners are clinically. . . through the heterodimerization of RXRs and PPARs that can bind to specific peroxisome proliferator response elements (PPREs). Both normal retinoid receptors and PML-RARalpha bind and activate the PPAR responsive element of the Acyl-CoA oxidase gene, indicating that retinoids and peroxisome proliferator receptors may share common target genes. As RXR is required for both RAR and PPAR to bind to the RARE and. . . the effect of X-RARalpha fusion proteins on the transactivating potential of RARalpha and PPARgamma in the presence or absence of ligand. Triplicate plates of COS7 cells were transiently transfected with four of the X-RARalpha fusion genes and reporter plasmids. The transactivation. . . different from the other APL fusion proteins as it results transcriptional superactivation at both RARE and PPRE upon treatment with ligand. By gel mobility shift assay we show that X-RARalpha fusion proteins bind differently to RARE and PPRE. Consistent with our. . . of NuMA-RARalpha into the myeloid leukemia cell line U937 reveals that these cells become resistant to the differentiative effects of vitamin D3. However, the response to TPA is intact, as examined by changes in cell surface markers (CD11b, CD14 and CD36).

L9 ANSWER 5 OF 13 MEDLINE DUPLICATE 4

ACCESSION NUMBER: 2001322651 MEDLINE

DOCUMENT NUMBER: 21131917 PubMed ID: 11237167

TITLE: Carotenoids and retinoids as suppressors on adipocyte

differentiation via nuclear receptors.

Kawada T; Kamei Y; Fujita A; Hida Y; Takahashi N; Sugimoto AUTHOR:

E; Fushiki T

Division of Applied Life Sciences, Graduate School of CORPORATE SOURCE:

Agriculture, Kyoto University, Japan.. fat@kais.kyoto-

BIOFACTORS, (2000) 13 (1-4) 103-9. SOURCE:

Journal code: 8807441. ISSN: 0951-6433.

PUB. COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

English LANGUAGE:

FILE SEGMENT: Priority Journals

200106 ENTRY MONTH:

Entered STN: 20010611 ENTRY DATE:

Last Updated on STN: 20010611 Entered Medline: 20010607

The adipocyte differentiation program is regulated by the sequential AB expression of transcriptional activators, mainly peroxisome proliferator activated receptor (PPAR) families. In the present study, we have decided to systematically examine the effects of vitamin A and its precursors, carotenoids and retinoids, on terminal differentiation from preadipocytes to adipocytes on the cellular and molecular aspects. The effects of active form of vitamin A, retinoic acid (RA), are believed to be mediated by specific nuclear receptor proteins [retinoic acid receptor (RAR)] which are members of the steroid and thyroid/retinoid receptor superfamily of ligand dependent transcriptional regulators, RARalpha, RARgamma, RXRalpha, and RXRbeta mRNA were abundant in adipose tissue and 3T3-L1 adipose cells. The autoregulated amplification of RARgamma mRNA was observed by these own ligands in 3T3-L1 cells. And, RA inhibited PPARgamma2 expression more effectively and caused concomitantly a greater inhibition of adipocyte differentiation. These. . . and retinoids are exhibited

through the RAR up-regulation and the suppression of PPARgamma2. The nature of the cross talk of vitamin A actions between the RARs,

RXRs and PPARs via co-activator in adipose tissue will likely prove to be

ANSWER 6 OF 13 MEDLINE DUPLICATE 5

ACCESSION NUMBER: 1999253823 MEDLINE

99253823 DOCUMENT NUMBER: PubMed ID: 10321906

Expression of the vitamin D receptor, of estrogen and TITLE: thyroid hormone receptor alpha- and beta-isoforms, and of

the androgen receptor in cultures of native mouse bone

marrow and of stromal/osteoblastic cells.

Gruber R; Czerwenka K; Wolf F; Ho G M; Willheim M; Peterlik **AUTHOR:**

> Department of General and Experimental Pathology, University of Vienna Medical School, Austria.

SOURCE: BONE, (1999 May) 24 (5) 465-73.

Journal code: 8504048. ISSN: 8756-3282.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

CORPORATE SOURCE:

important for.

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199907

Entered STN: 19990727 ENTRY DATE:

> Last Updated on STN: 19990727 Entered Medline: 19990714

AB Marrow stromal cells mediate the effect of lalpha, 25-dihydroxyvitamin D3 on formation of osteoclast-like cells from undifferentiated hematopoetic precursors in bone marrow. Induction by the vitamin D hormone of multinucleated, calcitonin receptor- and tartrate-resistant acid phosphatase-positive cells in primary mouse bone marrow culture can be modulated by other members of the steroid/thyroid. inhibitors of osteoclastogenesis. In an attempt to relate these

effects of the steroid/thyroid hormones to the presence of their respective nuclear receptors, we studied expression of the vitamin D receptor (VDR), estrogen receptor (ER) -alpha and -beta, thyroid hormone receptor (TR) -alpha and -beta, and androgen receptor (AR) in total bone marrow as well as primary marrow stromal cell cultures. By using reverse-transcriptase-polymerase chain reaction, in both. amplification products were obtained, which were identified by multiple restriction fragment length analysis as transcripts from mRNA specific for the ligand-binding domains of the VDR, ER-alpha, ER-beta, TR-alpha, TR-beta, and AR. Specific immunostaining by indirect peroxidase labeling revealed that among the various cell types present in bone marrow, the steroid/ thyroid hormone receptors are abundant particularly in marrow stromal cells. In another series of experiments, we extended our survey on receptor expression also to stromal/osteoblastic cell lines. At the mRNA level, the complete repertoire of steroid/thyroid hormone receptors was present in preadipocytic ST2 cells as well as in osteoblastic MC3T3-E1 cells. By immunocytochemical staining of the latter, it became apparent that single cells exhibit wide variations in intensity of specific signals for all the receptors investigated, so that, notably in contrast to primary stromal cells and ST2 cells, MC3T3-E1 display a mosaic pattern of receptor protein expression.

L9 ANSWER 7 OF 13 MEDLINE DUPLICATE 6

ACCESSION NUMBER: 1999191703 MEDLINE

DOCUMENT NUMBER: 99191703 PubMed ID: 10091603

TITLE: Stimulation of premature retinoic acid synthesis in Xenopus

embryos following premature expression of aldehyde

dehydrogenase ALDH1.

AUTHOR: Ang H L; Duester G

CORPORATE SOURCE: Gene Regulation Program, Burnham Institute, La Jolla, CA

92037, USA.

CONTRACT NUMBER: AA07261 (NIAAA)

SOURCE: EUROPEAN JOURNAL OF BIOCHEMISTRY, (1999 Feb) 260 (1)

227-34.

Journal code: 0107600. ISSN: 0014-2956. PUB. COUNTRY: GERMANY: Germany, Federal Republic of DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals
OTHER SOURCE: GENBANK-AF061833

ENTRY MONTH: 199904

ENTRY DATE: Entered STN: 19990504

Last Updated on STN: 19990504 Entered Medline: 19990422

AB In order for nuclear retinoic acid receptors to

mediate retinoid signaling, the ligand retinoic acid must first

be produced from its vitamin A precursor retinal.

Biochemical studies have shown that retinal can be metabolized in vitro to retinoic acid by members of the aldehyde. . .

L9 ANSWER 8 OF 13 MEDLINE DUPLICATE 7

ACCESSION NUMBER: 2000075981 MEDLINE

DOCUMENT NUMBER: 20075981 PubMed ID: 10609868

TITLE: Lipid soluble vitamins in gene regulation.

AUTHOR: Carlberg C

CORPORATE SOURCE: Institut fur Physiologische Chemie I, Heinrich-Heine-

Universitat, Dusseldorf, Germany.. carlberg@uni-

duesseldorf.de

SOURCE: BIOFACTORS, (1999) 10 (2-3) 91-7. Ref: 44

Journal code: 8807441. ISSN: 0951-6433.

PUB. COUNTRY: Netherlands

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LANGUAGE:

English

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

200002

ENTRY DATE:

Entered STN: 20000209

Last Updated on STN: 20000209

Entered Medline: 20000201

AB Vitamin A (retinol) and vitamin D are lipid soluble

vitamins that are precursors of the nuclear

hormones all-trans retinoic acid (RA) and lalpha, 25-dihydroxyvitamin D3

(VD) that bind with high affinity to their cognate nuclear

receptors, referred to as retinoic acid receptor (RAR)

and vitamin D receptor (VDR). Both types of

nuclear receptors are structurally related and belong to

the same subclass of the nuclear receptor superfamily,

a large family of **ligand**-inducible transcription factors. Both RAR and VDR form heterodimers preferentially with the **nuclear**

receptor for 9-cis RA, referred to as the retinoid X

receptor (RXR), but functional RAR-VDR heterodimers have also been

observed. Moreover, both types of nuclear receptors

interact in a ligand-dependent fashion with members of the same class of co-activator, co-repressor and co-integrator proteins. These

similar molecular mechanisms of action provide. . . can result in

either an additive or a transrepressive functional interference between RA and VD. The two remaining lipid soluble **vitamins**,

vitamins E and K, are not known to interact with nuclear

receptors, but their structure does not exclude this possibility.

Moreover, for vitamin E modulatory effects on transcription

factors, such as AP-1, have been described. This review will discuss

briefly gene regulation by the four lipid soluble vitamins.

L9 ANSWER 9 OF 13

MEDLINE

DUPLICATE 8

ACCESSION NUMBER:

1998151023 MEDLINE

DOCUMENT NUMBER:

98151023 PubMed ID: 9492059

TITLE:

Ligand-dependent regulation of retinoic acid receptor alpha

in rat testis: in vivo response to depletion and repletion

of vitamin A.

AUTHOR:

Akmal K M; Dufour J M; Vo M; Higginson S; Kim K H Department of Genetics and Cell Biology, Center for

CORPORATE SOURCE: Department of Genetics and Cell Biology, Center for

Reproductive Biology, Washington State University, Pullman

99164, USA.

SOURCE:

ENDOCRINOLOGY, (1998 Mar) 139 (3) 1239-48.

Journal code: 0375040. ISSN: 0013-7227.

PUB. COUNTRY:

United States

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE:

English

FILE SEGMENT:

Abridged Index Medicus Journals; Priority Journals

OTHER SOURCE:

GENBANK-U15211

ENTRY MONTH:

199803

ENTRY DATE:

Entered STN: 19980319

Last Updated on STN: 20000303

Entered Medline: 19980312

AB Male animals are sterile due to testicular degeneration in the absence of retinoic acid (RA) or functional retinoic acid receptor-alpha

(RAR alpha). This degeneration can be reversed by injecting retinol, a

precursor of RA, into vitamin A-deficient (VAD) rats. To
determine the relationship between this ligand-dependent

testicular degeneration and regeneration and the expression levels of RAR alpha messenger RNA and protein, testes were depleted and then. . .

advanced germ cells. Interestingly, the advanced germ cells still

contained RAR alpha, but the protein was primarily cytoplasmic instead of nuclear, indicating inactivity as a transcription factor. In VAD

testis, RAR alpha levels were low and then increased primarily in Sertoli.

DUPLICATE 9 ANSWER 10 OF 13 MEDLINE

ACCESSION NUMBER: 1998200046 MEDLINE

DOCUMENT NUMBER: 98200046 PubMed ID: 9540977

Keratinocyte differentiation is stimulated by activators of TITLE:

the nuclear hormone receptor PPARalpha.

Hanley K; Jiang Y; He S S; Friedman M; Elias P M; Bikle D **AUTHOR:**

D; Williams M L; Feingold K R

Department of Dermatology, University of California, San CORPORATE SOURCE:

Francisco, USA.

CONTRACT NUMBER: AR 39639 (NIAMS)

> AR29706 (NIAMS) HD 29706 (NICHD)

SOURCE: JOURNAL OF INVESTIGATIVE DERMATOLOGY, (1998 Apr) 110 (4)

368-75.

Journal code: 0426720. ISSN: 0022-202X.

PUB. COUNTRY:

United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

English LANGUAGE:

FILE SEGMENT: Priority Journals; Space Life Sciences

ENTRY MONTH: 199805

ENTRY DATE: Entered STN: 19980514

> Last Updated on STN: 20020124 Entered Medline: 19980504

AB Peroxisome proliferator activated receptors (PPAR) belong to the

superfamily of nuclear hormone receptors that

heterodimerize with the retinoid X receptor and regulate

transcription of several genes involved in lipid metabolism and adipocyte differentiation. Because of the role of 1,25-dihydroxyvitamin D3 and retinoic acid working through similar receptors (the

vitamin D receptor and retinoic acid receptor, respectively) on keratinocyte differentiation, we have examined the effects of activators of PPARalpha on keratinocyte differentiation. The . . low calcium (0.03 mM) and incubated in the rate of cornified. presence of clofibric acid, a potent PPARalpha activator. Involucrin, a cornified envelope precursor, and the cross-linking enzyme transglutaminase, were increased at both the message level (2-7-fold) and the protein level (4-12-fold) by clofibric. . . by itself induces keratinocyte differentiation. Finally, PPARalpha activators inhibit DNA synthesis. This study demonstrates that PPARalpha activators, including putative endogenous ligands such as fatty acids, induce

differentiation and inhibit proliferation in keratinocytes, and suggests a regulatory role for the PPARalpha in.

ANSWER 11 OF 13 MEDLINE **DUPLICATE 10**

ACCESSION NUMBER: 1998025946 MEDLINE

DOCUMENT NUMBER: 98025946 PubMed ID: 9379138

TITLE: The vitamin D hormone and its nuclear receptor: molecular

actions and disease states.

Haussler M R; Haussler C A; Jurutka P W; Thompson P D; AUTHOR:

Hsieh J C; Remus L S; Selznick S H; Whitfield G K

CORPORATE SOURCE: Department of Biochemistry, College of Medicine, University

of Arizona, Tucson 85724, USA.

CONTRACT NUMBER: AR 15781 (NIAMS)

> DK33351 (NIDDK) DK49604 (NIDDK)

JOURNAL OF ENDOCRINOLOGY, (1997 Sep) 154 Suppl S57-73. SOURCE:

Journal code: 0375363. ISSN: 0022-0795.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, ACADEMIC)

LANGUAGE: English

FILE SEGMENT: Priority Journals; Space Life Sciences ENTRY MONTH:

199711

ENTRY DATE:

Entered STN: 19971224

Last Updated on STN: 19971224 Entered Medline: 19971112

Vitamin D plays a major role in bone mineral homeostasis by AR promoting the transport of calcium and phosphate to ensure that. these ions are sufficient for the normal mineralization of type I collagen matrix in the skeleton. In contrast to classic vitamin D-deficiency rickets, a number of vitamin D-resistant rachitic syndromes are caused by acquired and hereditary defects in the metabolic activation of the vitamin to its hormonal form, 1,25-dihydroxyvitamin D3 (1,25(OH)2D3), or in the subsequent functions of the hormone in target cells. The actions of 1,25(OH)2D3 are mediated by the nuclear vitamin D receptor (VDR), a phosphoprotein which binds the hormone with-high affinity and regulates the expression of genes via zinc finger-mediated DNA binding and protein-protein interactions. In hereditary hypocalcemic vitamin D-resistant rickets (HVDRR), natural mutations in human VDR that confer patients with tissue insensitivity to 1,25(OH)2D3 are particularly instructive in revealing VDR structure function relationships. These mutations fall into three categories: (i) DNA binding/nuclear localization, (ii) hormone binding and (iii) heterodimerization with retinoid X receptors (RXRs). That all three classes of VDR mutations generate the HVDRR phenotype is consistent with a basic model of the active receptor as a DNA-bound, 1,25(OH)2D3-liganded heterodimer of VDR and RXR. Vitamin D responsive elements (VDREs) consisting of direct hexanucleotide repeats with a spacer of three nucleotides have been identified in the promoter regions of positively controlled genes expressed in bone, such as osteocalcin, osteopontin, beta 3-integrin and vitamin D 24-OHase. The 1,25(OH)2D3 ligand promotes VDR-RXR heterodimerization and specific, high affinity VDRE binding, whereas the ligand for RXR, 9-cis retinoic acid (9-cis RA), is capable of suppressing 1,25(OH)2D3-stimulated transcription by diverting RXR to form homodimers. However, . . . the AF-2 domains participate neither in VDR-RXR heterodimerization nor in TFIIB association, it is hypothesized that they contact, in a ligand-dependent fashion, transcriptional coactivators such as those of the steroid receptor coactivator family, constituting yet a third protein-protein interaction for VDR. Therefore, in VDR-mediated transcriptional activation, 1,25(OH)2D3 binding to VDR alters the conformation of the ligand binding domain such that it: (i) engages in strong heterodimerization with RXR to facilitate VDRE binding, (ii) influences the RXR ligand binding domain such that it is resistant to the binding of 9-cis RA but active in recruiting coactivator . to attract TFIIB and the balance of the RNA polymerase II transcription machinery, culminating in repeated transcriptional initiation of VDRE-containing, vitamin D target genes. Such a model would explain the action of 1,25(OH)2D3 to elicit bone remodeling by stimulating osteoblast and osteoclast precursor gene expression, while concomitantly triggering the termination of its hormonal signal by inducing the 24-OHase catabolizing enzyme.

L9 ANSWER 12 OF 13 MEDLINE DUPLICATE 11

ACCESSION NUMBER: 96209825 MEDLINE

DOCUMENT NUMBER: 96209825 PubMed ID: 8643496

TITLE: Novel retinoic acid receptor ligands in Xenopus embryos.

AUTHOR: Blumberg B; Bolado J Jr; Derguini F; Craig A G; Moreno T A;

Chakravarti D; Heyman R A; Buck J; Evans R M

CORPORATE SOURCE: Gene Expression Laboratory, Salk Institute for Biological

Studies, La Jolla, CA 92037, USA.

CONTRACT NUMBER: CA54418 (NCI)

DK48022 (NIDDK) HD27183 (NICHD)

SOURCE: PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE

UNITED STATES OF AMERICA, (1996 May 14) 93 (10) 4873-8.

Journal code: 7505876. ISSN: 0027-8424.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199607

Entered STN: 19960726 ENTRY DATE:

> Last Updated on STN: 19960911 Entered Medline: 19960718

Retinoids are a large family of natural and synthetic compounds related to AB vitamin A that have pleiotropic effects on body physiology, reproduction, immunity, and embryonic development. The diverse activities of retinoids are primarily mediated by two families of nuclear retinoic acid receptors, the RARs and RXRs. Retinoic acids are thought to be the only natural ligands for these receptors and are widely assumed to be the active principle of vitamin A. However, during an unbiased, bioactivity-guided fractionation of Xenopus embryos, we were unable to detect significant levels of all-trans or. . . is capable of binding to and transactivating RARs. In addition to its inherent activity, 4-oxoretinaldehyde appears to be a metabolic precursor of two other RAR ligands, 4-oxoretinoic acid and 4-oxoretinol. The remarkable increase in activity of retinaldehyde and retinol as a consequence of 4-oxo derivatization suggests.

ANSWER 13 OF 13 MEDLINE

ACCESSION NUMBER: 95012031 MEDLINE

PubMed ID: 7927175 DOCUMENT NUMBER: 95012031

TITLE:

An overexpression of retinoic acid receptor alpha blocks

myeloid cell differentiation at the promyelocyte stage.

AUTHOR: Onodera M

Department of Pediatrics, Hokkaido University School of CORPORATE SOURCE:

Medicine, Sapporo, Japan.

HOKKAIDO IGAKU ZASSHI. HOKKAIDO JOURNAL OF MEDICAL SCIENCE, SOURCE:

(1994 May) 69 (3) 466-75, 477.

Journal code: 17410290R. ISSN: 0367-6102.

PUB. COUNTRY: Japan

Journal; Article; (JOURNAL ARTICLE) DOCUMENT TYPE:

LANGUAGE: Japanese

Priority Journals FILE SEGMENT:

ENTRY MONTH: 199410

Entered STN: 19941222 ENTRY DATE:

> Last Updated on STN: 19941222 Entered Medline: 19941026

Retinoic acid (RA), a vitamin A derivative, exerts a wide range AB of biological effects related to cell proliferation and differentiation. The pleiotropic effects of RA are thought to be mediated through specific nuclear RA receptors (RARs). RARs are members of the steroid/thyroid hormone receptor superfamily and exhibit a molecular structure that possess discrete DNA-binding and RA (ligand) -binding domains. In hematopoietic system, RA and RARs, predominantly RAR alpha may play key roles for the proliferation and . . is effective to suppress myeloid cell differentiation of. differentiation and RAR alpha plays a crucial role in the terminal differentiation of myeloid precursors. The system described here may serve as a model for studying the the essential genes for differentiation of normal bone.

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			DERWENT	
-	9	(nuclear adj receptor) same (reporter adj gene) and cloning and cell and	USPAT;	2002/04/16 15:01
	1	screen?	US-PGPUB;	
			EPO; JPO;	
			DERWENT	
•	49	(nuclear adj receptor) same (reporter adj gene)	USPAT;	2002/04/16 15:02
			US-PGPUB;	
			EPO; JPO;	
			DERWENT	